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Accurate Noninvasive Estimation of Pulmonary Vascular Resistance by Doppler Echocardiography in Patients With Chronic Heart Failure

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OBJECTIVES This study was undertaken to explore further the relationship between Doppler-derived parameters of pulmonary flow and pulmonary vascular resistance (PVR) and to determine whether PVR could be accurately estimated noninvasively from Doppler flow velocity measurements in patients with chronic heart failure.

BACKGROUND The assessment of PVR is of great importance in the management of patients with heart failure. However, because of the inconclusive and conflicting data available, Doppler estimation of PVR is still considered unreliable.

METHODS Simultaneous Doppler echocardiographic examination and right heart catheterization were performed in 63 consecutive sinus rhythm heart failure patients with severe left ventricular systolic dysfunction. Hemodynamic PVR was calculated with the standard formula. The following Doppler variables on pulmonary flow and tricuspid regurgitation velocity curve were correlated with PVR: maximal systolic flow velocity, pre-ejection period (PEP), acceleration time (AcT), ejection time, total systolic time (TT), velocity time integral, and right atrium-ventricular gradient.

RESULTS At univariate analysis, all variables except maximal systolic flow velocity and velocity time integral showed a significant, although weak, correlation with PVR. The best correlation found was between AcT and PVR ($r = -0.68$). By regression analysis, only PEP, AcT and TT entered into the final equation, with a cumulative $r = 0.87$. When the function (PEP/AcT)/TT was correlated with PVR, the correlation coefficient further improved to 0.96. Of note, this function prospectively predicted PVR ($r = 0.94$) after effective unloading manipulations.

CONCLUSIONS The analysis of Doppler-derived pulmonary systolic flow is a reliable and accurate tool for estimating and monitoring PVR in patients with chronic heart failure due to left ventricular systolic dysfunction. (J Am Coll Cardiol 2001;37:1813–9) © 2001 by the American College of Cardiology

Assessment of pulmonary vascular resistance (PVR) is of great importance in the management of patients with chronic heart failure, and it is an essential component of orthotopic heart transplantation recipient evaluation. Currently, this information is obtained only with invasive cardiac catheterization. An accurate noninvasive measurement of PVR would prove helpful in eliminating the risks, costs and discomfort associated with cardiac catheterization.

In patients with heart failure, Doppler echocardiography has become routine in the bedside noninvasive estimation of several hemodynamic variables (1–9). However, because of the inconclusive and conflicting available data, Doppler estimation of PVR is still considered unreliable (10,11). Nevertheless, in patients with chronic heart failure, particularly those with advanced heart failure requiring serial right heart catheterization for hemodynamic suitability for heart transplantation, a noninvasive mode of estimating PVR would be a highly desirable alternative.

We hypothesized a strict relationship between Doppler systolic pulmonary flow and PVR. The present study was undertaken to explore further the relationship between Doppler-derived parameters of pulmonary flow and PVR and to determine whether PVR could be accurately estimated noninvasively from Doppler flow velocity measurements in patients with chronic heart failure.

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METHODS

Study population. This prospective study included 63 consecutive sinus rhythm patients (55 men; mean age [± SD] 57 ± 8 years) with chronic heart failure and severe left ventricular (LV) systolic dysfunction (as defined by echocardiographic ejection fraction ≤35%) undergoing diagnostic right heart catheterization. Informed consent was obtained, and the study was approved by the local Ethics Committee at Veruno Medical Center.

Study protocol. After baseline echocardiographic evaluation, patients underwent simultaneous hemodynamic and echocardiographic examinations.

ECHOCARDIOGRAPHY. A Hewlett-Packard (Andover, Massachusetts) Sonos 1500 ultrasound system equipped with 2.5- and 3.5-MHz probes was used. Echocardiographic studies were performed in left lateral decubitus or supine position; all echocardiographic images were stored

Abbreviations and Acronyms

AcT = acceleration time
CO = cardiac output
ECG = electrocardiogram
EjT = ejection time
LV = left ventricular
PADP = pulmonary artery diastolic pressure
PAMP = pulmonary artery mean pressure
PASP = pulmonary artery systolic pressure
PCWP = pulmonary capillary wedge pressure
PEP = pre-ejection period
PVR = pulmonary vascular resistance
SD = standard deviation
TT = total systolic time

on super-VHS videotape. Left ventricular volumes were calculated from orthogonal apical views using the biplane area-length method. Ejection fraction was derived from the standard equation. Both mitral and tricuspid regurgitation were detected and graded using color flow Doppler, according to previously reported criteria that took into account both the width and depth of regurgitant jets in relation to the size of the receiving chamber from multiple views (12) and the size of the jet at the regurgitant orifice (13).

A pulsed-wave Doppler recording of pulmonary artery flow was analyzed in a transverse parasternal view at the level of great vessels. The sample volume was positioned in the right ventricular outflow tract just below the pulmonary plane. Care was taken to align the sample volume and the axis of the bloodstream correctly to obtain the highest possible Doppler velocity signal with the smallest amount of spectral dispersion and to avoid possible noises due to the Swan-Ganz catheter. Tricuspid regurgitation was detected by continuous-wave Doppler velocity curve from apical views. Gain and filters of the machine were adjusted to define precisely the onset and the end of both systolic pulmonary profile and tricuspid regurgitant velocity curve.

SIMULTANEOUS HEMODYNAMIC AND DOPPLER EXAMINATION. All patients were studied in the fasting state. A 7F Swan-Ganz catheter (Baxter Healthcare, Edwards Critical Care Division, Deerfield, Illinois) was introduced using the Seldinger technique through a femoral or right internal jugular vein and positioned under fluoroscopic guidance in a pulmonary artery. After a 10-min rest for stabilization, pulmonary artery systolic pressure (PASP), pulmonary artery diastolic pressure (PADP) and pulmonary capillary wedge pressure (PCWP) were obtained at end-expiration. The wedged position of the tip of the right heart catheter during recording of the occlusion waveform was verified fluoroscopically. Pulmonary artery mean pressure (PAMP) was obtained by using the standard formula:

$$\text{PAMP} = [\text{PADP} + {}^{1}/_{3}\,(\text{PASP} - \text{PADP})]$$

Cardiac output (CO) was determined by the thermodilution method as the mean of three consecutive measurements not varying by >10%. The PVR, expressed in Wood units, was calculated as:

$$\text{PVR} = (\text{PAMP} - \text{PCWP})/\text{CO}$$

Hemodynamic and Doppler parameters were recorded simultaneously. A custom-designed system connected the echocardiographic unit to computerized hemodynamic instruments so that it was possible to record Doppler and hemodynamic traces and electrocardiogram (ECG) on-line onto both videotape and a strip chart recorder at 100 mm/s for off-line analysis. Immediately after pulmonary systolic flow velocity recording, continuous-wave spectral Doppler images of the tricuspid regurgitation were recorded; thereafter, hemodynamic PAMP and PCWP were obtained. To avoid the interference of injection of saline bolus on the Doppler signal recording, CO was determined when Doppler measurements were completed. On the systolic pattern of pulmonary flow, the following variables were considered: maximal systolic flow velocity, systolic velocity time integral, acceleration time (AcT), expressed as the time interval between the onset of ejection to the time of peak flow velocity, and ejection time (EjT), expressed as the time interval between the onset and end of the systolic flow velocity recording.

On the continuous-wave spectral Doppler velocity curve of tricuspid regurgitation, the onset of regurgitation was identified (Fig. 1, top) and extrapolated from the QRS signal (used as a reference point) to the zero line on an isorhythmic recording interval of Doppler pulmonary flow (Fig. 1, bottom). Functional pre-ejection period (PEP) was defined as the distance between the onset of tricuspid regurgitation and the onset of pulmonary systolic flow. In the presence of elevated PASP and PVR, tricuspid regurgitation may well continue after the premature closure of the pulmonary valve (i.e., the premature completion of the ejection period); thus, we calculated the total systolic time (TT) as the summation of PEP and EjT rather than as the total duration of tricuspid regurgitation (Fig. 1). An average of five beats was analyzed. Finally, peak velocity on the tricuspid regurgitation velocity curve was measured, and the Bernoulli equation was used to calculate the right atrium-ventricular gradient.

After completion of baseline measurements, 15 patients with elevated PVR underwent a nitroprusside test performed as follows: nitroprusside was infused at an initial dose of 0.5 μg/kg body weight. The dose was increased by 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ up to a maximum of 4 $\mu g \cdot kg^{-1} \cdot min^{-1}$ unless the patient developed hypotension (systolic blood pressure <85 mm Hg), bradycardia (heart rate <50 beats/min), or a drop of PCWP to <12 mm Hg. At the end of the test, simultaneous hemodynamic and Doppler recordings were repeated.

REPRODUCIBILITY OF NONINVASIVE DOPPLER VARIABLES. Both intraobserver and interobserver reproducibility of Doppler echocardiographic measurements were assessed in

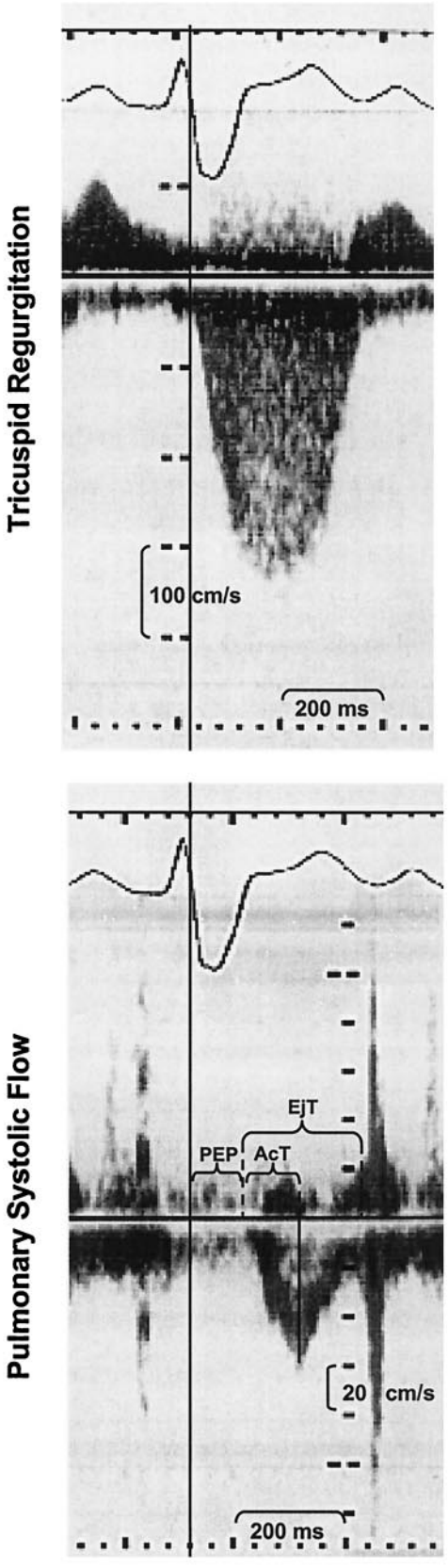

Figure 1. Continuous-wave Doppler recording of tricuspid regurgitant flow **(top)** and pulsed-wave recording of pulmonary flow **(bottom)** in the same patient. AcT = acceleration time; EjT = ejection time; PEP = pre-ejection period.

Table 1. Clinical and Hemodynamic Characteristics of the Study Patients

	All Patients (n = 63) Mean ± SD	Range
Age (yrs)	57 ± 8	37–75
Coronary artery disease (%)	35 (36%)	
Heart rate (beats/min)	80 ± 14	55–111
PVR (Wood)	2.5 ± 1.5	0.2–9.2
CO (liter/min)	4.5 ± 1.1	2.1–8
PCWP (mm Hg)	18 ± 9	3–37
PAMP (mm Hg)	28 ± 12	8–55

Data presented are mean values ± SD or number of patients (%).
CO = cardiac output; PAMP = pulmonary artery mean pressure; PCWP = pulmonary capillary wedge pressure; PVR = pulmonary vascular resistance.

11 consecutive patients. Measurements were repeated by the same observer after an interval of at least one week and by a second independent observer. The variability of the estimated PVR was then evaluated by calculating the mean relative difference between paired measurements and its standard deviation (SD).

Statistical analysis. Results are given as mean ± 1 SD. The Doppler variables were compared with the hemodynamic PVR variables by simple linear regression analysis. Stepwise regression analysis was also used to evaluate the predictive power of independent variables. Noninvasive PVR was compared with hemodynamic PVR by using linear regression analysis and calculating the mean relative difference between paired measurements and their SD (14). A p value <0.05 was considered statistically significant.

RESULTS

Clinical, Doppler echocardiographic and hemodynamic data at baseline are presented in Tables 1 and 2. All patients had moderate to severe LV dilation and severe systolic dysfunction (mean ejection fraction 17 ± 5%). Moderate to severe tricuspid regurgitation was detected in about 50% of patients (severe regurgitation in 16%), whereas moderate to severe mitral regurgitation was present in most of the patients (90%), being severe in 32%. Mean values of PVR and heart rate were 2.5 ± 1.5 Wood units (range 0.2 to 9.2 Wood) and 80 ± 14 beats/min, respectively.

Relation of Doppler echocardiographic variables to PVR. Adequate recordings of the tricuspid regurgitation velocity curve were obtained in 46 of the 63 study patients; recordings of the onset of tricuspid regurgitation and systolic pulmonary flow patterns were obtained in all patients. Correlations between Doppler flow velocity variables and PVR are reported in Table 3. No correlation was found between PVR and maximal pulmonary systolic flow velocity, and a weak correlation existed between PVR and systolic velocity time integral ($r = -0.39$), PEP ($r = 0.44$) and EjT ($r = -0.32$). The AcT ($r = -0.68$) and TT ($r = -0.63$) on pulmonary systolic flow pattern and right atrium-ventricular gradient ($r = 0.58$) showed the highest, although modest, correlations with PVR. When all Doppler variables were

Table 2. Two-Dimensional and Doppler Echocardiographic Parameters

	Mean ± SD	Range
Two-dimensional data		
Ejection fraction (%)	17 ± 5	9–33
LVEDVI (ml/m^2)	156 ± 43	86–262
RVEDD (mm)	46 ± 9	25–72
Right atrial area (cm^2)	22 ± 7	10–36
Left atrial area (cm^2)	29 ± 10	10–44
TR mild/moderate/severe	32/21/10	
MR mild/moderate/severe	16/27/20	
Doppler pulmonary flow		
Vmax (cm/s)	72 ± 18	35–118
VTI (s)	11.2 ± 3.5	3.6–20.4
PEP (s)	0.065 ± 0.025	0.019–0.133
AcT (s)	0.088 ± 0.026	0.037–0.179
EjT (s)	0.247 ± 0.043	0.135–0.354
TT (s)	0.312 ± 0.049	0.169–0.437
(PEP/AcT)/TT	2.6 ± 1.4	0.6–7.3
Tricuspid regurgitation velocity curve		
Right A-V gradient (mm Hg)	43 ± 14	20–70

Data presented are mean values ± SD or number of patients.
AcT = acceleration time; A-V = atrium-ventricular; EjT = ejection time; LVEDVI = left ventricular end-diastolic volume index; MR = mitral regurgitation; PEP = pre-ejection period; RVEDD = right ventricular end-diastolic diameter; TR = tricuspid regurgitation; TT = total systolic time; Vmax = maximal systolic velocity; VTI = velocity time integral.

analyzed in a stepwise forward regression analysis, only AcT, PEP and TT entered into the final equation for predicting PVR (Table 4). The most important determinant was AcT, whereas PEP and TT provided a smaller contribution. The cumulative correlation coefficient (r) was 0.87.

Based on this analysis and on pathophysiologic considerations, we hypothesized that the PEP/AcT ratio, normalized for TT, would represent the best mathematic function for predicting PVR. In fact, when the function (PEP/AcT)/TT was correlated to PVR, the correlation coefficient (r) further improved to 0.96 (Fig. 2). The analysis led to the following equation:

$$PVR = -0.156 + 1.154 * [(PEP/AcT)/TT]$$

On Bland-Altman analysis, the mean relative difference between measured and estimated PVR was close to 0 (0.56) for the whole population, indicating the absence of any systematic error.

Table 3. Correlations Between Pulmonary Doppler Variables and Pulmonary Vascular Resistance

	Correlation Coefficient	p Value
Vmax	0.16	0.38
VTI	−0.39	0.02
PEP	0.44	0.002
AcT	−0.68	0.0001
EjT	−0.32	0.01
TT	−0.63	0.01
Right A-V gradient	0.58	0.01

Abbreviations as in Table 2.

Table 4. Results of Stepwise Regression Analysis of Doppler Indexes on Pulmonary Flow Versus Pulmonary Vascular Resistance

Step No.	Cumulative r Value	F Value to Enter	No. of Variables Included
1. AcT	0.68*	52	1
2. PEP	0.84*	15	2
3. TT	0.87*	39	3

Final equation: PVR = 5.8 − 31.7 * AcT + 29.9 * PEP − 9.9 * TT. *$p < 0.0001$.
Abbreviations as in Table 2.

Doppler-derived pulmonary systolic flow changes after unloading manipulations. The effects of unloading manipulations were simultaneously evaluated by hemodynamic and Doppler measurements in 15 patients with elevated baseline PVR. Changes in pulmonary flow pattern variables and PVR after nitroprusside infusion are presented in Table 5. Mean PVR decreased significantly from 4.2 ± 1.3 to 2.2 ± Wood units. A significant reduction in PEP ($p < 0.05$), together with a significant increase ($p < 0.001$) in AcT, with no significant changes in EjT and TT of pulmonary systolic flow, was found (Table 5). When the reliability of the equation reported above was prospectively tested in these patients, baseline values and changes of PVR after unloading were still accurately predicted (Fig. 3).

Reproducibility of noninvasive estimation of PVR by Doppler echocardiography. Both intra- and interobserver variability for different sets of measurements of Doppler-derived variables of pulmonary systolic flow were 5% and 4% for maximal systolic velocity, 4.8% and 4% for velocity time integral, 3% and 2.5% for PEP, 1.2% and 1.4% for AcT and 4% and 3.5% for EjT. Coefficients of inter- and intraobserver variability were not statistically significant for any of the tricuspid flow variables measured.

DISCUSSION

Doppler echocardiography provides a simple and noninvasive means of assessing and monitoring several hemodynamic variables in different cardiac conditions. Evaluation of PVR is crucial in the assessment of patients with chronic heart failure and is of paramount importance in potential candidates for heart transplantation. So far, however, PVR has shown a poor correlation with Doppler variables. In this study we demonstrated that PVR can be reliably estimated in heart failure patients by combining Doppler echocardiographic variables of pulmonary flow: the PEP/AcT ratio, normalized for TT, allows an accurate quantitative estimation of PVR. Because this relation appears linear between 0 and 9 Wood units (Fig. 2), it may be used to estimate the absolute PVR between these values. Importantly, when the equation was prospectively tested in the subgroup of patients with elevated PVR undergoing unloading manipulations (i.e., nitroprusside infusion), the correlation still remained excellent, indicating that the method can easily allow for an identification of patients with an adverse

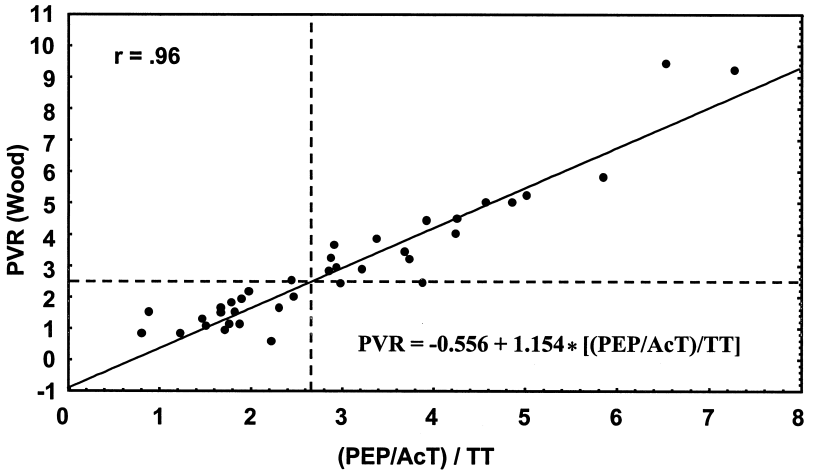

Figure 2. Scatterplot of linear correlation between measured and Doppler-determined pulmonary vascular resistance (PVR). **Vertical dashed line** marks the value of 2.6 in Doppler-determined PVR that predicts a value of 2.5 Wood in measured PVR **(horizontal dashed line),** above which PVR is commonly considered to be increased. AcT = acceleration time; PEP = pre-ejection period; TT = total systolic time, all of them on Doppler pulmonary flow.

pulmonary vascular hemodynamic status and the determination of hemodynamic suitability for heart transplantation.

Comparison with other studies. Invasive determination of PVR is the standard of care: PAMP minus left atrial pressure divided by CO, expressed in Wood units, is the well-accepted formula to calculate PVR. Although Doppler echocardiography is an excellent methodologic alternative to right heart catheterization, given the complexity of the parameter to be measured, it is obvious that a single Doppler variable would not permit a precise estimation of PVR. Previous studies (10,11) correlating hemodynamic variables with Doppler-derived pulmonary flow velocity measurements in small groups of patients with a wide range of cardiac diseases found significant correlations between total pulmonary resistance and AcT, or AcT index (AcT/right ventricular EjT), but the correlation with PVR was poor. Invasive and noninvasive data sets, however, were not acquired simultaneously, and this represents a major limitation of these earlier studies.

In addition, because of the considerable scatter about the regression line, this technique does not allow a precise estimate of PVR, being useful only in grouping patients with normal and elevated PVR. To overcome these limitations, Stein et al. (15) recently derived PVR noninvasively by calculating first the transpulmonary gradient as mean PAMP minus left atrial pressure, and then dividing the transpulmonary gradient by CO. Even in this study (Stein et al. [15]), however, data sets were not acquired simultaneously, and the noninvasive technique was fairly laborious.

Table 5. Hemodynamic Pulmonary Vascular Resistance and Pulmonary Doppler Variables at Baseline and After Nitroprusside Infusion

	Baseline	Nitroprusside Infusion
PVR (Wood)	4.2 ± 1.3	2.2 ± 1.1
Vmax (cm/s)	84 ± 18	86 ± 26
VTI (s)	9.3 ± 2.4	11.2 ± 2.2
PEP (s)	0.084 ± 0.030	0.064 ± 0.019*
AcT (s)	0.075 ± 0.022	0.093 ± 0.019†
EjT (s)	0.221 ± 0.061	0.240 ± 0.039
TT (s)	0.306 ± 0.082	0.303 ± 0.032

*$p < 0.05$. †$p < 0.001$.
Abbreviations as in Tables 1 and 2.

In our study, even with Doppler and hemodynamic data sets simultaneously recorded, the correlation between each single Doppler parameter and PVR was weak. In particular, although the right atrium-ventricular gradient was strongly related to PASP (r = 0.92) and PAMP (r = 0.86), its correlation with PVR was weak (r = 0.58). On the contrary, a simple function (PEP/AcT)/TT derived from the Doppler pulmonary flow profile allowed for a quantitatively reliable estimation of PVR.

Pathophysiologic considerations. Based on the well-known correlation between Doppler pulmonary systolic flow and pulmonary artery pressures, we hypothesized that a reliable estimation of PVR could be possible through an accurate analysis and interpretation of Doppler pulmonary systolic velocity profile. As expected, we found a poor correlation between each single Doppler variable and PVR, whereas a stepwise regression analysis identified AcT, PEP and TT as independent predictors of PVR. We then elaborated further on these three variables and their relation. As PVR increases, the right ventricle has to generate a higher pressure to exceed the forces opposing the pulmonary valve opening; consequently, a prolonged PEP should be found.

Despite normal PVR, however, a prolonged PEP could also be found in the presence of right ventricular dysfunction or intraventricular conduction disturbances. This is why in our study the onset of PEP was not traditionally measured on the ECG QRS peak. We instead measured PEP as the time between the continuous-wave, Doppler-derived onset of tricuspid regurgitation and the onset of Doppler pulmonary systolic velocity flow, as the onset of tricuspid regurgitation represents the very beginning of intraventricular pressure rise, when ventricular contraction

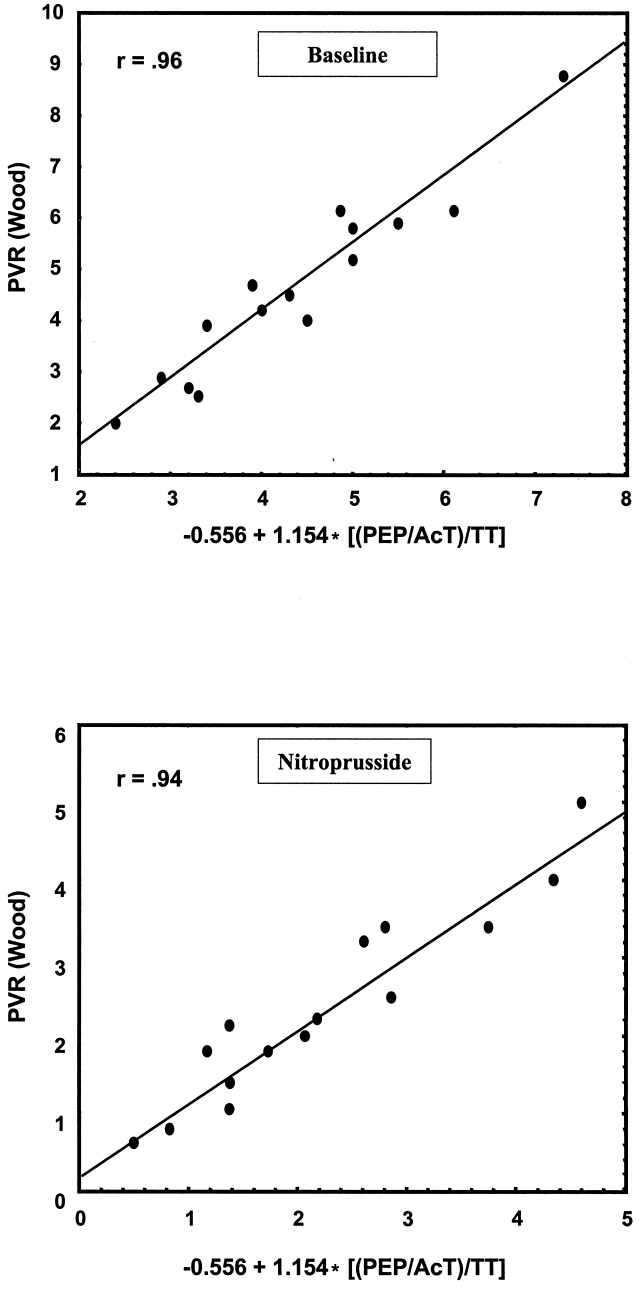

Figure 3. Scatterplots of Doppler-determined versus measured pulmonary vascular resistance (PVR) values both before **(top)** and after **(bottom)** unloading manipulations (nitroprusside infusion) in a group of patients (n = 15) with baseline elevated PVR. AcT = acceleration time; PEP = pre-ejection period; TT = total systolic time.

becomes effective and consequently able to generate the flow through the pulmonary valve.

Furthermore, in the presence of elevated PVR, after pulmonary valve opening, right ventricular pressure will rapidly equalize pulmonary artery pressure. As a consequence, a shorter AcT, together with a shorter EjT, should be expected. In light of these considerations, we hypothesized that the ratio between these different times would better express the pathophysiologic relation between Doppler variables and PVR. Indeed, the PEP/AcT ratio, normalized for TT, showed the strongest relation to PVR (r = 0.96).

Study limitations. The major limitation of this study could be the need to perform measurements in two different sets of Doppler recordings—that is, during tricuspid regurgitation and during systolic pulmonary flow, to measure PEP; it should also be noted, however, that the hemodynamic assessment of PVR requires three separate data set recordings (i.e., PAMP, PCWP, CO). The accurate selection of iso-rhythmic cycles to avoid possible mistakes in PEP evaluation is crucial. In this view, patients with atrial fibrillation were excluded, although theoretically it should not pose any absolute limitation if more cardiac cycles are averaged. Further studies are warranted not only in this subset of patients but also in other patient groups—those with primary pulmonary vascular disease and those with normal ejection fractions.

Invasive CO was obtained by the thermodilution method. Although the Fick equation should be recommended when assessing CO in patients with heart failure, particularly when severe tricuspid regurgitation is present, thermodilution is typically what is performed clinically.

In our study population of heart failure patients, at least mild tricuspid regurgitation was present. The presence of trivial tricuspid regurgitation could affect the recording of the continuous-wave Doppler profile of the regurgitant flow for PEP estimation, but it should be emphasized that only the very onset of tricuspid regurgitation is required, usually easy to obtain even in trivial insufficiency.

Conclusions. Our study provides evidence that the analysis of Doppler-derived pulmonary systolic flow is a reliable tool for estimating and monitoring PVR in patients with chronic heart failure. This noninvasive methodology may constitute an alternative to routine right heart catheterization in potential heart transplantation candidates and could help physicians in the safe and cost-effective bedside management of patients with congestive heart failure.

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